CASE REPORT JSLS

Recurrent Asystolic Cardiac Arrest and Laparoscopic Cholecystectomy: A Case Report and Review of the Literature

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ABSTRACT

Laparoscopic surgery has become a durable alternative for both gynecologic and general surgical procedures, but reported complications are increasing. We describe the case of a 70-year-old male undergoing routine laparoscopic cholecystectomy for gallstone pancreatitis who developed asystolic cardiac arrest intraoperatively. A review of the literature revealed 2 cases of asystolic cardiac arrest during laparoscopy: one was during laparoscopic cholecystectomy and one was during diagnostic laparoscopy for gynecologic evaluation.

Key Words: Asystole, Laparoscopic cholecystectomy, Laparoscopy, Cardiac arrest.

INTRODUCTION

Laparoscopic surgery has established itself as a durable alternative for both gynecologic and general surgical procedures. With increasing popularity and greater utility, the types and number of reported complications are increasing. We describe the case of a 70-year-old male undergoing routine laparoscopic cholecystectomy for gallstone pancreatitis who developed asystolic cardiac arrest intraoperatively. A review of the literature revealed 2 cases of asystolic cardiac arrest during laparoscopy: one was during laparoscopic cholecystectomy and one was during diagnostic laparoscopy for gynecologic evaluation.

CASE REPORT

A 70-year-old male with a past medical history negative for ischemic heart disease with asymptomatic myocardial infarction was admitted to the hospital with a 24-hour history of severe, acute epigastric abdominal pain. His medications on admission consisted of aspirin alone. His social history was significant for 2 pack per day tobacco use.

Physical examination revealed a well-developed elderly gentleman in no apparent distress. He was afebrile with a pulse rate of 85 beats/minute. Cardiopulmonary evaluation revealed a regular rhythm with no evidence of gallop or murmurs. His point of maximal impulse was nondisplaced, and he had no evidence of jugular venous distension. His lung fields were clear on auscultation bilaterally with no evidence of congestion. His abdomen was soft with no masses, hernias, or palpable evidence of organomegaly. He demonstrated mild to moderate tenderness to palpation of his epigastrium and right upper abdominal quadrant. He did not have a Murphy sign or any peritoneal findings. Initial investigations included the following tests: complete blood count: white blood cell count 14.6; hemoglobin/hematocrit 15.1/44.5, platelets 399 000; basic metabolic profile: Na 138, K 4.1, Cl 103, CO_2 26, urea 14, creatinine 0.9, glucose 138; liver function tests: total bilirubin 0.8, albumin 3.1, aspartate aminotransferase (AST) 576, alanine aminotransferase (ALT) 417, alkaline phosphatase 352, LDH 1639; amylase 1173; lipase 5339. Initial electrocardiography revealed regular rhythm with first-degree AV block

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and no evidence of acute coronary ischemia. A chest x-ray showed mild cardiomegaly, hyperinflated lungs, and no evidence of infiltrate or congestion. Ultrasonography of the gallbladder showed several gallstones with a common bile duct measurement to 8 mm, no wall thickness, and no evidence of pericholecystic fluid. Computer tomography of the abdomen illustrated diffuse evidence of nonhemorrhagic acute pancreatitis.

The patient was admitted to the hospital with a diagnosis of gallstone pancreatitis. He was not allowed to take anything by mouth and was started on empiric intravenous antibiotics for presumed persistent common bile duct obstruction. Over ensuing days, the patient's laboratory evaluations showed a continuously decreasing amylase and lipase until normality was achieved on hospital day number 4. It was deemed that the patient would require endoscopic retrograde cholangiopancreatography (ERCP) and cardiology clearance prior to undergoing laparoscopic cholecystectomy. Because the patient's acute symptomology had entirely resolved and he was tolerating a regular diet without recurrence of pain, he was discharged home with plans to undergo ERCP and cardiac evaluation as an outpatient. ERCP was attempted by a well-experienced gastroenterologist; cannulation of the sphincter of Oddi was unsuccessful secondary to a duodenal diverticulum, and the procedure was aborted. Cardiac assessment consisting of a stress test was performed, which was negative for unstable coronary circulation.

The patient returned to the hospital 6 weeks after the initial evaluation for laparoscopic cholecystectomy. In the interim, he had been doing well, tolerating a low-fat diet, and had stopped smoking 2 weeks prior to the scheduled surgery. He had been on a nicotine patch for this duration. The patient had stopped taking aspirin 10 days prior to surgery.

Preoperative medication comprised Mefoxin and was discontinued approximately 30 minutes prior to the scheduled surgery. In the operating room, the patient was preoxygenated with 80% oxygen to achieve a peripheral pulse oxygen saturation of 100%. He was subsequently induced with etomidate and succinylcholine and then intubated and ventilated. Appropriate intubation was confirmed by positive end-tidal CO_2 and auscultation of administered breaths in bilateral lung fields. Anesthesia was maintained with nitrous oxide and fentanyl. In the supine position, access into the peritoneal cavity was attained using the open Hassan technique via an intraumbilical incision. The opening intraperitoneal pressure was 8 mm Hg. Pneumoperitoneum was initially achieved to an intraperitoneal pressure of 15 mm Hg by first administering low-flow CO_2 followed by high-flow CO_2. Our right subxiphoid and right subcostal trocars were then inserted without incident. The gallbladder was then grasped with a gallbladder grasper and retracted in the lateral/cephalad direction for approximately 15 seconds resulting in an asystolic episode for 30 seconds. The gallbladder was immediately released and pneumoperitoneum evacuated. The patient was administered atropine, with subsequent resumption of sinus cardiac rhythm. With resumption of stable vital signs, reinsufflation of the peritoneal cavity to 15 mm Hg was again performed using low-flow CO_2 administration. Upon initial grasping of the gallbladder, the patient once again became asystolic. Sinus rhythm was achieved within 5 seconds of releasing the gallbladder and relieving the pneumoperitoneum. A third attempt at gallbladder manipulation was made without insufflation but with direct visualization of the gallbladder. Again, the patient became asystolic after 2 to 3 seconds of retraction. Cardiac rhythm resumed approximately 5 seconds after the gallbladder was released. Throughout all 3 episodes, adequacy of ventilation was confirmed bilaterally, oxygen saturation remained at 100%, capnometry readings of 35 to 40 mm Hg were obtained, and the patient remained normothermic. No acute electrocardiographic changes were appreciated with resumption of cardiac rhythm in the operating room. It was decided to abort further attempts at cholecystectomy at this time. The patient was extubated in the operating room, transferred to the postanesthesia recovery room, and maintained on telemetry.

Postoperative cardiology and electrophysiology evaluation did not reveal a primary cardiac event as the cause of the patient's asystolic episodes. Monitoring over the next 24 hours did not reveal any further arrhythmias. Additionally, the patient's cardiac isoenzyme profile over the 24-hour period was negative for acute myocardial injury. Given these findings coupled with the patient's respiratory stability intraoperatively, it was deemed that a severe vagal reaction in response to gallbladder retraction was the source of the patient's asystolic episodes. Therefore, the patient underwent insertion of a temporary pacemaker prior to a reattempt at a laparoscopic cholecystectomy. The pacer was set to a rate greater than

the patient's resting heart rate. The second procedure was performed using a pneumoperitoneum of 15 mm Hg and was accomplished without incident. The pacemaker was removed in the early postoperative period, and the patient was discharged home without further incident.

DISCUSSION

Hemodynamically significant cardiovascular phenomena are a known and real complication of laparoscopic surgery. The causes of cardiovascular collapse during laparoscopy include CO_2 pulmonary embolization, cardiac arrhythmias, vagal reactions secondary to peritoneal distention during insufflation or viscus manipulation, and diminished cardiac preload secondary to caval compression. Asystolic cardiac arrest is a potential manifestation of these hemodynamically significant events. In a review of the literature, the American Association of Gynecology reports an incidence of one in 2500 cases of asystolic arrest during laparoscopy.[1] We were able to identify only one previously described case of asystole during laparoscopic cholecystectomy.[2]

Several possible causes exist for cardiovascular collapse during laparoscopy. One possibility that is frequently discussed is CO_2 gas embolization. In such instances, the clinical manifestation generally includes a diminished end-tidal CO_2, tachycardia, diminished breath sounds in a specific lung field on auscultation, and a classic cardiac murmur associated with gas embolization. The general mechanism is perceived to be infiltration of insufflated CO_2 into venous/lymphatic channels with subsequent pulmonary migration. It is unlikely that the asystolic arrest in our case is secondary to gas embolization because our patient failed to exhibit any of the above signs.

An alternative cause of hemodynamically significant cardiovascular changes during laparoscopy is hypoxia or hypercapnia resulting in cardiac arrhythmias. It is believed that the combination of the Trendelenburg positioning and elevated intraabdominal compartment pressures predispose a patient to aspiration, resulting in hypoxia and possibly hypercapnia. Theoretically, hypercapnia may also occur from CO_2 absorption during pneumoperitoneum. However, it is unlikely that clinically significant elevations in CO_2 levels on blood-gas measurements can be detected.[1]

Elevated intraabdominal pressures can diminish venous return to the heart, preload, resulting in diminished cardiac output. With intraabdominal pressures ranging from 12 to 15 mm Hg, a slight decrease occurs in preload without a significant alteration in cardiac output. However, with intraabdominal pressures in excess of 40 mm Hg, a significant caval compression leads to a decreased preload and cardiac output.[3] Kelman et al[4] theorize that 2 counter-balancing mechanisms occur during peritoneal insufflation. The first is an increase in central blood volume due to the forcing of blood out of the splanchnic circulation. The second is a diminished preload secondary to peripheral pooling of blood in the lower extremities in combination with the reversed Trendelenburg position. The result is a temporary increase in circulating blood volume followed by a sustained decrease in central pressures.[4]

Pneumothorax, pneumomediastinum, or both may also complicate general laparoscopy. These typically occur in patients with primary pulmonary pathology, such as pulmonary/mediastinal blebs, which rupture under positive pressure. Alternatively, these complications may arise secondary to excessive inspiratory airway pressures. Lehman et al[3] delineate the possibility of tension pneumothorax during insufflation secondary to a congenital diaphragmatic defect. Cases of pneumothorax/pneumomediastinum generally present with hypotension, tachycardia, diminished breath sounds in a lung field, and possibly subcutaneous emphysema.

Shifren et al1 describe a case of asystolic cardiac arrest during gynecologic laparoscopy that is attributed to rapid peritoneal distension during insufflation. Under circumstances of elevated intraabdominal pressures, it is postulated that manipulation of certain pelvic structures/organs may further elevate intraabdominal pressures. We do not believe this to be the cause in our case. Even during gallbladder handling without pneumoperitoneum, asystole occurred.[1] It is our opinion that the asystolic cardiac arrest in our case was secondary to a severe vagal reaction that was triggered by manipulation of the gallbladder. This was validated by the fact that recurrent asystole was documented for approximately 5 seconds upon grasping the gallbladder without elevated intraabdominal pressures. Sinus rhythm resumed only after the gallbladder was released in all 3 instances. Additionally, no reproducible hemodynamic sequelae occurred during the successful attempt at laparoscopic cholecystectomy, once the temporary pacemaker was in place. We could not identify any clinical criteria, including altered CO_2

levels, hypoxia, diminished breath sounds, or tachycardia to suggest any of the other proposed mechanisms of cardiovascular collapse during laparoscopy that are described above.

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